# Correspondence

## More on *Mycoplasma pneumoniae* Pneumonia

TO THE EDITOR: Chan and Welsh's and Cassell's recent comprehensive reviews of fulminant *Mycoplasma pneumoniae* pneumonia raised issues as to the pathogenesis of severe respiratory insufficiency and the value of corticosteroid therapy.[1,2] Our experience with a patient with *M pneumoniae* pneumonia invites further investigation of its molecular pathobiology and of the value of corticosteroid therapy.

*Report of a Case*

The patient, a 38-year-old female schoolteacher who did not smoke, had severe headache, fever, sore throat, nonproductive cough, and progressive dyspnea develop a week before hospital admission. Notably abnormal laboratory findings at the time of admission included a leukocyte count of $24 \times 10^9$ per liter (24,000 per $mm^3$) with a leftward shift; a platelet count of $120 \times 10^9$ per liter (120,000 per $mm^3$); a hemoglobin level of 98 grams per liter (9.8 grams per dl); and a hematocrit of 0.30 (30%). Serum sodium was 130 mmol per liter (130 mEq per liter); lactate dehydrogenase, 535 U; $Po_2$, 44.9 mm of mercury; $Pco_2$, 45.9 mm of mercury; and pH 7.35. A chest x-ray film showed bipulmonary acinar infiltrates. Antimicrobial therapy that was initiated consisted of intravenous vancomycin hydrochloride, 1 gram every 12 hours; aztreonam, 2 grams every 8 hours; metronidazole, 500 mg every 6 hours; erythromycin, 1 gram every 6 hours; and ribavirin, 33 mg per kg as a single loading dose, with 16 mg every 6 hours for 4 days and 8 mg per kg every 8 hours for 3 days. The ribavirin was added because of a recent exposure to rodents.

Despite the therapeutic regimen and 100% oxygen by mask, she continued to deteriorate, and intubation was necessary. Systemic vascular resistance ranged between 350 and 700 dynes per cm, the cardiac output from 6 to 8 liters per minute, and persistent hypotension required vasopressor support. Disseminated intravascular coagulation (DIC) supervened with a prolonged prothrombin time and partial thromboplastin time, increased fibrin-split products and D-dimer, and thrombocytopenia. Her arterial $Po_2$ was maintained with a fraction of inspired oxygen of 60% to 100% and positive end-expiratory pressure.

On the sixth hospital day, immunoglobulin (Ig) M antibodies to *M pneumoniae* were reported at 1:64 by immunofluorescence, and a test for IgG was negative. By this time, all sputum and blood cultures failed to yield a pathogen or were sterile, and serologic tests for *Legionella* and *Chlamydia* species, coccidioidomycosis, histoplasmosis, Q fever, influenza, parainfluenza, adenovirus, the human immunodeficiency virus, Sin Nombre, Puumala, and Hantaan viruses were negative or insubstantially present, and a purified-protein derivative (PPD) and urine test for *Legionella* species antigen were negative. A perfusion lung scan revealed a low likelihood of pulmonary emboli. All antimicrobial agents were discontinued, and the patient received intravenous doxycycline, 100 mg every 12 hours, and methylprednisolone sodium succinate, 60 mg every 12 hours.

The patient quickly became afebrile, her chest x-ray film showed improvement, the hemodynamic instability and DIC resolved, and the nasotracheal tube was removed four days later.

*Discussion*

We interpret these events to confirm the fulminant pneumonia as caused by *M pneumoniae* accompanied by hemodynamic and hematologic changes that closely resemble sepsis. We would postulate that a superantigen of *M pneumoniae*, as described by Cole for *Mycoplasma arthritidis*,[3] caused a massive T-cell proliferation and release of proinflammatory cytokines. These initiated the extensive pulmonary infiltrates and hemodynamic abnormalities noted in this patient. The dramatic response from corticosteroid therapy likely resulted from its T-lymphocytolytic properties by apoptosis.

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**REFERENCES**

1. Chan ED, Welsh CH: Fulminant *Mycoplasma pneumoniae* pneumonia. West J Med 1995; 162:133-142

2. Cassell GH: Severe mycoplasma disease—Rare or underdiagnosed? West J Med 1995; 162:172-175

3. Cole BC: The immunobiology of *Mycoplasma arthritidis* and its superantigen MAM. Curr Top Microbiol Immunol 1991; 174:107-119

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TO THE EDITOR: Chan and Welsh, in their recent review of fulminant *Mycoplasma pneumoniae* pneumonia, suggest that an enhanced host cellular immune response may account for the progression of illness in severely ill patients.[1] In addition, they comment that the use of systemic steroids "may be salutary." I present a case of severe *M pneumoniae* pneumonia that followed a biphasic course in which there was an initial response to antibiotics, but where the use of parenteral steroids ultimately proved to be lifesaving.

*Report of a Case*

The patient, a 41-year-old previously healthy female physician in active practice, was admitted to the hospital with dry cough, fever, and dehydration. Her illness had begun several weeks before admission when a dry cough developed in her and other family members. Her cough became productive 12 days before admission, and she had chills, malaise, and fever. The patient self-